

WEST Search History

DATE: Thursday, August 03, 2006

Hide?	<u>Set Name</u>	<u>Query</u>	<u>Hit Count</u>
	<i>DB=PGPB,USPT,JPAB,DWPI; PLUR=YES; OP=ADJ</i>		
<input type="checkbox"/>	L2	L1 and bone	15
<input type="checkbox"/>	L1	zmax1 or zmax 1	26

END OF SEARCH HISTORY

\$\$*STN;HighlightOn= ***;HighlightOff=*** ;

Connecting via Winsock to STN

Welcome to STN International! Enter x:x

LOGINID:ssspla1633cxq

PASSWORD:

TERMINAL (ENTER 1, 2, 3, OR ?):2

***** Welcome to STN International *****

NEWS 1 Web Page URLs for STN Seminar Schedule - N. America

NEWS 2 "Ask CAS" for self-help around the clock

NEWS 3 FEB 27 New STN AnaVist pricing effective March 1, 2006

NEWS 4 APR 04 STN AnaVist \$500 visualization usage credit offered

NEWS 5 MAY 10 CA/CAPLUS enhanced with 1900-1906 U.S. patent records

NEWS 6 MAY 11 KOREAPAT updates resume

NEWS 7 MAY 19 Derwent World Patents Index to be reloaded and enhanced

NEWS 8 MAY 30 IPC 8 Rolled-up Core codes added to CA/CAPLUS and

USPATFULL/USPAT2

NEWS 9 MAY 30 The F-Term thesaurus is now available in CA/CAPLUS

NEWS 10 JUN 02 The first reclassification of IPC codes now complete in

INPADOC

NEWS 11 JUN 26 TULSA/TULSA2 reloaded and enhanced with new search and

and display fields

NEWS 12 JUN 28 Price changes in full-text patent databases EPFULL and PCTFULL

NEWS 13 JUL 11 CHEMSAFE reloaded and enhanced

NEWS 14 JUL 14 FSTA enhanced with Japanese patents

NEWS 15 JUL 19 Coverage of Research Disclosure reinstated in DWPI

NEWS EXPRESS JUNE 30 CURRENT WINDOWS VERSION IS V8.01b, CURRENT

MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP), AND CURRENT DISCOVER FILE IS DATED 26 JUNE

2006.

NEWS HOURS STN Operating Hours Plus Help Desk Availability

NEWS LOGIN Welcome Banner and News Items

NEWS IPC8 For general information regarding STN

implementation of IPC 8

NEWS X25 X.25 communication option no longer available

Enter NEWS followed by the item number or name to see news on that specific topic.

All use of STN is subject to the provisions of the STN Customer agreement. Please note that this agreement limits use to scientific research. Use for software development or design or

implementation of commercial gateways or other similar uses is prohibited and may

result in loss of user privileges and other penalties.

***** STN Columbus *****

FILE 'HOME' ENTERED AT 16:07:06 ON 03 AUG 2006

=> FIL EMBASE BIOSIS CAPLUS

COST IN U.S. DOLLARS SINCE FILE TOTAL

FULL ESTIMATED COST ENTRY SESSION 0.21 0.21

FILE 'EMBASE' ENTERED AT 16:07:17 ON 03 AUG 2006

Copyright (c) 2006 Elsevier B.V. All rights reserved.

FILE 'BIOSIS' ENTERED AT 16:07:17 ON 03 AUG 2006

Copyright (c) 2006 The Thomson Corporation

FILE 'CAPLUS' ENTERED AT 16:07:17 ON 03 AUG 2006

USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER

AGREEMENT.

PLEASE SEE "HELP USAGETERMS" FOR DETAILS.

COPYRIGHT (C) 2006 AMERICAN CHEMICAL SOCIETY (ACS)

=> s zmax 1 or zmax1

L1 53 ZMAX 1 OR ZMAX1

=> s l1 and bone

L2 8 L1 AND BONE

=> dup rem l2

PROCESSING COMPLETED FOR L2

L3 7 DUP REM L2 (1 DUPLICATE REMOVED)

=> d bib abs 1-

YOU HAVE REQUESTED DATA FROM 7 ANSWERS - CONTINUE? Y/(N):y

L3 ANSWER 1 OF 7 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

DUPLICATE 1

AN 2004:383095 BIOSIS <<LOGINID::20060803>>

DN PREV200400388096

TI High ***bone*** mass gene of 1.1q13.3.

AU Carulli, John P. [Inventor, Reprint Author]; Little, Randall D.

[Inventor]; Recker, Robert R. [Inventor]; Johnson, Mark L.

[Inventor]

CS ASSIGNEE: Genome Therapeutics Corporation

PI US 6780609 20040824

SO Official Gazette of the United States Patent and Trademark Office Patents,

(Aug 24 2004) Vol. 1285, No. 4.

<http://www.uspto.gov/web/menu/patdata.html>

e-file.

ISSN: 0098-1133 (ISSN print).

DT Patent

LA English

ED Entered STN: 29 Sep 2004

Last Updated on STN: 29 Sep 2004

AB The present invention relates to methods and materials used to isolate and

detect a high ***bone*** mass gene and a corresponding wild-type gene,

and mutants thereof. The present invention also relates to the high

bone mass gene, the corresponding wild-type gene, and mutants

thereof. The genes identified in the present invention are implicated in

bone development. The invention also provides nucleic acids,

including coding sequences, oligonucleotide primers and probes, proteins,

cloning vectors, expression vectors, transformed hosts, methods of

developing pharmaceutical compositions, methods of identifying molecules

involved in ***bone*** development, and methods of diagnosing and

treating diseases involved in ***bone*** development. In preferred

embodiments, the present invention is directed to methods for treating,

diagnosing and preventing osteoporosis.

L3 ANSWER 2 OF 7 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

AN 2004:351355 BIOSIS <<LOGINID::20060803>>

DN PREV200400354810

TI High ***bone*** mass gene of 11q13.3.

AU Carulli, John P. [Inventor, Reprint Author]; Little, Randall D.

[Inventor]; Recker, Robert R. [Inventor]; Johnson, Mark L.

[Inventor]

CS Southboro, MA, USA

ASSIGNEE: Genome Therapeutics Corporation; Creighton University School of

Medicine, Omaha, NE, USA

PI US 6770461 20040803

SO Official Gazette of the United States Patent and Trademark Office Patents,

(Aug 3 2004) Vol. 1285, No. 1.

<http://www.uspto.gov/web/menu/patdata.html>

e-file.

ISSN: 0098-1133 (ISSN print).

DT Patent

LA English

ED Entered STN: 26 Aug 2004

Last Updated on STN: 26 Aug 2004

AB The present invention relates to methods and materials used to isolate and

detect a high ***bone*** mass gene and a corresponding wild-type gene,

and mutants thereof. The present invention also relates to the high

bone mass gene, the corresponding wild-type gene, and mutants

thereof The genes identified in the present invention are implicated in

bone development. The invention also provides nucleic acids, including coding sequences, oligonucleotide primers and probes, proteins, cloning vectors, expression vectors, transformed hosts, methods of developing pharmaceutical compositions, methods of identifying molecules involved in ***bone*** development, and methods of diagnosing and treating diseases involved in ***bone*** development. In preferred embodiments, the present invention is directed to methods for diagnosing and preventing osteoporosis.

L3 ANSWER 3 OF 7 CAPLUS COPYRIGHT 2006 ACS on STN AN 2002:888494 CAPLUS <<LOGINID::20060803>>
DN 137:381503
T1 Compositions and methods for modulating Dkk-mediated protein interactions and their diagnostic and therapeutic uses
IN Allen, Kristina; Anisowicz, Anthony; Bhat, Bheem M.; Damagnez, Veronique; Robinson, John Allen; Yaworsky, Paul J.
PA Genome Therapeutics Corporation, USA; Wyeth, John and Brother Ltd.
SO PCT Int. Appl., 376 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 4
PATENT NO. KIND DATE APPLICATION NO.
DATE

PI WO 2002092015 A2 20021121 WO 2002-US15982 20020517
WO 2002092015 A3 20031023
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
CA 2446582 AA 20021121 CA 2002-2446582 20020517
EP 1395285 A2 20040310 EP 2002-744162 20020517
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR
BR 200209836 A 20041207 BR 2002-9836 20020517
JP 2005512508 T2 20050512 JP 2002-588934 20020517
US 2004038860 A1 20040226 US 2002-182936 20020802
PRAI US 2001-291311P P 20010517
US 2002-353058P P 20020201
US 2002-361293P P 20020304
WO 2002-US15982 W 20020517

AB The present invention provides reagents, compds., compns., and methods relating to interactions of the extracellular domain of LRP5/ ***ZMax1***, HBM (a variant of LRP5), and/or LRP6 with Dkk, including Dkk-1. The various nucleic acids, polypeptides, antibodies, assay methods, diagnostic methods, and methods of treatment of the present invention are related to and impact on Dkk, LRP5, LRP6, HBM, and Wnt signaling. The invention claims sequences for peptide aptamers which bind to LRP5 or Dkk-1 and sequences for Dkk-1 peptides which are recognized by antibodies. HBM is a Gly171Val polymorphism in LDL receptor-related protein LRP5/Zmax, which has been identified as conferring a high ***bone*** mass phenotype in

a population of related humans. The protein dickkopf-1 (Dkk-1) is required for head formation in early development and murine limb morphogenesis and is reported to be an antagonist of the Wnt signaling pathway. Dkk-1 protein interacts with the ligand-binding domain of LRP5. Dkk-1 also binds to LRP6, but the EGF repeat domains of LRP6 rather than the ligand-binding domain are required for interaction. Dkk-1 is able to repress LRP5-mediated Wnt signaling but not HBM-mediated Wnt signaling and Dkk-1 also inhibits LRP6 activity. LRP5, LRP6, HBM, Dkk and Wnt are implicated in ***bone*** and lipid cellular signaling. Thus, the present invention provides reagents and methods for modulating lipid levels and/or ***bone*** mass and is useful in the treatment and diagnosis of abnormal lipid levels and ***bone*** mass disorders, such as osteoporosis. Examples of the invention include a yeast two-hybrid screen for Dkk-1 interacting proteins, generation of LRP5 polymorphism-specific antibodies and Dkk-1 specific antibodies, effects of exogenous Dkk-1 on Wnt-mediated signaling in the Xenopus embryo assay, and effects of recombinant Dkk and Wnt3a/1 on TCF-luciferase reporter gene expression in human cell lines with endogenous LRP5/6.

L3 ANSWER 4 OF 7 CAPLUS COPYRIGHT 2006 ACS on STN AN 2002:888480 CAPLUS <<LOGINID::20060803>>
DN 137:380994
T1 High ***bone*** mass variants of the human ***Zmax1*** /LRP5 gene modulate ***bone*** mass and lipid levels
IN Allen, Kristina; Anisowicz, Anthony; Graham, James R.; Morales, Arturo; Yaworsky, Paul J.; Liu, Wei
PA Genome Therapeutics Corporation, USA; Wyeth, John and Brother Ltd.
SO PCT Int. Appl., 629 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 4
PATENT NO. KIND DATE APPLICATION NO.
DATE

PI WO 2002092000 A2 20021121 WO 2002-US14877 20020513
WO 2002092000 A3 20041007
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
CA 2446821 AA 20021121 CA 2002-2446821 20020513
BR 2002009563 A 20041207 BR 2002-9563 20020513
EP 1483288 A2 20041208 EP 2002-746370 20020513
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR
JP 2004537289 T2 20041216 JP 2002-588919 20020513
US 2005070699 A1 20050331 US 2004-477173 20041104
PRAI US 2001-290071P P 20010511
US 2001-291311P P 20010517
US 2002-353058P P 20020201
US 2002-361293P P 20020304
WO 2002-US14877 W 20020513
AB The present invention relates to methods and materials used to express an

HBM-like polypeptide derived from HBM (high ***bone*** mass), LRP5 or LRP6 in animal cells and transgenic animals. The HBM gene comprises 23 exons on human chromosome 11q13.3, and is shown to be an allele of the ***Zmax1*** /LRP5 gene; a variety addnl. single nucleotide polymorphisms are also identified. The ***Zmax1*** (LRP5) protein with a glycine-170-valine substitution causes a HBM phenotype involving high ***bone*** mass and modified lipid levels, whereas the valine-170 isoform does not. This mutation is in the propeller 1 domain of the protein, and modulates Wnt signaling, Dkk activity, and/or LRP5/6 activity. The present invention also relates to transgenic animals expressing the HBM-like polypeptides. The invention provides nucleic acids, including coding sequences, oligonucleotide primers and probes, proteins, cloning vectors, expression vectors, transformed hosts, methods of developing pharmaceutical compns., methods of identifying mols. involved in ***bone*** development, and methods of diagnosing and treating diseases involved in ***bone*** development and lipid modulation. In preferred embodiments, the present invention is directed to methods for treating, diagnosing and preventing osteoporosis.

L3 ANSWER 5 OF 7 CAPLUS COPYRIGHT 2006 ACS on STN AN 2001:886643 CAPLUS <<LOGINID::20060803>> DN 136:32816

TI Regulating lipid levels via the human ***Zmax1*** or high-***bone***-mass HBM gene
IN Carulli, John P.; Little, Randall D.; Recker, Robert R.; Johnson, Mark L.

PA Genome Therapeutics Corporation, USA; Creighton University School of Medicine

SO PCT Int. Appl., 409 pp.

CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 4

PATENT NO.	KIND	DATE	APPLICATION NO.
PI WO 2001092891	A2	20011206	WO 2001-US16946

DATE

PI WO 2001092891	A2	20011206	WO 2001-US16946
------------------	----	----------	-----------------

20010525

WO 2001092891	A3	20020725	
---------------	----	----------	--

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ,

CA, CH, CN,

CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB,

GD, GE, GH,

GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC,

LK, LR,

LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO,

NZ, PL, PT,

RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA,

UG, UZ,

VN, YU, ZA, ZW

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT,

BE, CH, CY,

DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE,

TR, BF,

BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD,

TG

CA 2410253	AA	20011206	CA 2001-2410253
------------	----	----------	-----------------

20010525

AU 2001269712	A2	20011211	AU 2001-269712
---------------	----	----------	----------------

20010525

EP 1285002	A2	20030226	EP 2001-948240
------------	----	----------	----------------

20010525

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE,

MC, PT,

IE, SI, LT, LV, FI, RO, MK, CY, AL, TR

BR 2001011057	A	20030415	BR 2001-11057
---------------	---	----------	---------------

20010525

JP 2004523724	T2	20040805	JP 2002-501047
---------------	----	----------	----------------

20010525

NZ 522600	A	20040924	NZ 2001-522600
-----------	---	----------	----------------

20010525

ZA 2002008977	A	20031105	ZA 2002-8977
---------------	---	----------	--------------

20021105

PRAI US 2000-578900	A	20000526	
---------------------	---	----------	--

WO 2001-US16946	W	20010525	
-----------------	---	----------	--

AB The present invention relates to the high ***bone*** mass

(HBM) gene,

the corresponding wild-type gene (***Zmax1***), and mutants thereof.

The ***Zmax1*** /HBM gene was located on chromosome 11q13.3 by genetic linkage and mutation anal. Cloning methods using bacterial

artificial chromosomes enabled focus on the chromosome region of 11q13.3 and sequencing of the autosomal dominant gene. A guanine-to-thymine

polymorphism at position 582 (glycine-to-valine at position 171 in the protein)in ***Zmax1*** gene produces the HBM gene and the HBM

phenotype as well as altered lipid levels. Hybridization for ***Zmax1*** is primarily detected in areas of ***bone***

involved in remodeling, including the endosteum and trabecular ***bone*** within

the metaphysis; pos. signals are also obsd in selected ***bone***

lining cells of the periosteum and epiphysis and in chondrocytes within the growth plate. The genes identified in the present invention are

implicated in regulation of physiol. lipid levels, and thereby lipid-mediated diseases and conditions. The invention also provides

nucleic acids, including coding sequences, oligonucleotide primers and probes, proteins, cloning vectors, expression vectors,

transformed hosts, methods of developing pharmaceutical compns., methods of identifying mols.

involved in lipid level regulation in a subject. In preferred embodiments, the present invention is directed to methods for treating and

preventing atherosclerosis, arteriosclerosis cardiovascular disease, atherosclerotic and arteriosclerotic assocd. conditions.

L3 ANSWER 6 OF 7 CAPLUS COPYRIGHT 2006 ACS on STN

AN 2001:763189 CAPLUS <<LOGINID::20060803>>

DN 135:328141

TI Human gene ***Zmax1*** of 11q13.3, HBM (high ***bone***

mass)

allele, encoded polypeptides, and their diagnostic and

therapeutic uses

IN Carulli, John P.; Little, Randall D.; Recker, Robert R.; Johnson, Mark L.

PA Genome Therapeutics Corporation, USA

SO PCT Int. Appl., 443 pp.

CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 4

PATENT NO.	KIND	DATE	APPLICATION NO.
PI WO 2001077327	A1	20011018	WO 2000-US16951

DATE

PI WO 2001077327	A1	20011018	WO 2000-US16951
------------------	----	----------	-----------------

20000621

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ,

CA, CH, CN,

CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE,

GH, GM, HR,

HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS,

LT,

LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL,

PT, RO, RU,

SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US,

UZ, VN,

YU, ZA, ZW

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT,

BE, CH, CY,

DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE,

BF, BJ,

CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

US 6770461	B1	20040803	US 2000-544398
------------	----	----------	----------------

20000405

US 6780609	B1	20040824	US 2000-543771
------------	----	----------	----------------

20000405

CA 2402410	AA	20011018	CA 2000-2402410
------------	----	----------	-----------------

20000621

EP 1268775	A1	20030102	EP 2000-941578
------------	----	----------	----------------

20000621

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE,

MC, PT,

IE, SI, LT, LV, FI, RO, MK, CY, AL

JP 2004515209	T2	20040527	JP 2001-575181
---------------	----	----------	----------------

20000621

NZ 521769	A	20041224	NZ 2000-521769
-----------	---	----------	----------------

20000621

US 2003219793 A1 20031127 US 2003-374979
 20030228
 PRAI US 2000-543771 A 20000405
 US 2000-544398 A 20000405
 US 1998-71449P P 19980113
 US 1998-105511P P 19981023
 US 1999-229319 A2 19990113
 US 2000-578900 W 20000526
 WO 2000-US16951 W 20000621
 US 2002-240851 A1 20021004

AB The present invention relates to methods and materials used to isolate and detect a high ***bone*** mass gene and a corresponding wild-type gene, and mutants thereof. The present invention also relates to the high ***bone*** mass allele, the corresponding wild-type gene, ***Zmax1***, and mutants thereof. The genes identified in the present invention are implicated in ***bone*** development and in focal adhesion signaling. The invention also provides nucleic acids, including coding sequences, oligonucleotide primers and probes, proteins, cloning vectors, expression vectors, transformed hosts, methods of developing pharmaceutical compns., methods of identifying mols. involved in ***bone*** development, and methods of diagnosing and treating diseases involved in ***bone*** development. In preferred embodiments, the present invention is directed to methods for treating, diagnosing and preventing osteoporosis. The invention describes expanded pedigree anal. and genetic linkage anal. of a high ***bone*** mass (HBM) gene now known as an allele of human gene ***Zmax1***. Older individuals with the HBM allele do not show loss of ***bone*** mass compared to normal individuals, do not have osteoporosis, and do not have any known high ***bone*** mass syndrome. Gene ***Zmax1*** was localized between genetic markers on human chromosome 11q13.3 and subsequently, BAC clones with the gene were sequenced. The HBM allele is inherited as an autosomal dominant gene and is a G.fwdarw. T mutation at nucleotide 582 in exon 3 which results in a G171V substitution in the encoded protein. Addnl. genotyping of 911 individuals established that the HBM allele is rare and never found in unaffected individuals. "Silent" SNPs (single nucleotide polymorphisms) in the gene ***Zmax1*** region were also identified. Gene ***Zmax1*** encodes an LDL-receptor-related protein and the HBM mutation occurs in a conserved region of the presumed extracellular domain. Proteins interacting with the cytoplasmic domain of gene ***Zmax1*** protein in a yeast two-hybrid assay were identified and include many proteins found at cell-cell and cell-matrix contact sites. These results suggest a potential role for gene ***Zmax1*** in focal adhesion signaling and suggest that regulating gene ***Zmax1*** expression or protein binding may affect ***bone*** processes.

RE.CNT 12 THERE ARE 12 CITED REFERENCES AVAILABLE FOR THIS RECORD
 ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 7 OF 7 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
 AN 1986:319794 BIOSIS <<LOGINID::20060803>>
 DN PREV198682044099; BA82:44099
 TI ASSIGNMENT OF THE GENE FOR DYSKERATOSIS CONGENITA TO XQ28.
 AU CONNOR J M [Reprint author]; GATHERER D; GRAY F C; PIRRI L A; AFFARA N A
 CS UNIVERSITY DEPARTMENT OF GENETICS, DUNCAN GUTHRIE INSTITUTE OF MEDICAL GENETICS, YORKHILL, GLASGOW, G3 9SJ, UK
 SO Human Genetics, (1986) Vol. 72, No. 4, pp. 348-351.
 CODEN: HUGEDQ. ISSN: 0340-6717.
 DT Article
 FS BA

LA ENGLISH
 ED Entered STN: 8 Aug 1986
 Last Updated on STN: 8 Aug 1986
 AB Dyskeratosis congenita is an X-linked recessive disorder with diagnostic dermatological features, ***bone*** marrow hypofunction, and a predisposition to neoplasia in early adult life. Linkage analysis was undertaken in an extensive family with the condition using the Xg blood group and 17 cloned X chromosomal DNA sequences which recognise restriction fragment length polymorphisms (RFLPs). No recombination was observed between the locus for dyskeratosis congenita (DKC) and the RFLPs identified by DXS52 (St 14-1) (Zmax = 3.33 at .THETA.max = 0 with 95% confidence limits of 0 to 14 cM). Similarly no recombination was observed for the disease locus and F8 (***Zmax*** = ***1*** .23 at .THETA.max = 0) nor for DXS15 (***Zmax*** = ***1*** .62 at .THETA.max = 0), but both of these markers were only informative in part of the family whereas DXS52 was fully informative. DXS52, DXS15, and F8 are known to be tightly linked and have previously been assigned to Xq28. Thus the gene for dyskeratosis congenita can be assigned to Xq28. These DNA sequence polymorphisms will be of clinical value for carrier detection and prenatal diagnosis.

=> s carulli, j7/au
 L4 85 CARULLI, J7/AU

=> s Little, r7/au
 L5 2133 LITTLE, R7/AU

=> s recker, r7/au
 L6 674 RECKER, R7/AU

=> s johnson, m7/au
 L7 20657 JOHNSON, M7/AU

=> s l4 or l5 or l7 or l6
 L8 23482 L4 OR L5 OR L7 OR L6

=> s l8 and (zmax or hbm)
 L9 20 L8 AND (ZMAX OR HBM)

=> dup rem l9
 PROCESSING COMPLETED FOR L9
 L10 11 DUP REM L9 (9 DUPLICATES REMOVED)

=> d bib abs 1-
 YOU HAVE REQUESTED DATA FROM 11 ANSWERS -
 CONTINUE? Y(N):y

L10 ANSWER 1 OF 11 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN DUPLICATE 1
 AN 2006250138 EMBASE <<LOGINID::20060803>>
 TI LRP5: Structural and molecular aspects.
 AU ***Johnson M.L.***
 CS Dr. M.L. Johnson, Department of Oral Biology, UMKC School of Dentistry, 650 East 25th Street, Kansas City, MO 64108, United States. johnsonmark@umkc.edu
 SO Clinical Reviews in Bone and Mineral Metabolism, (2006) Vol. 4, No. 2, pp. 97-106.
 Refs: 67
 ISSN: 1534-8644 CODEN: CRBMBF
 CY United States
 DT Journal; General Review
 FS 022 Human Genetics
 030 Pharmacology
 033 Orthopedic Surgery
 037 Drug Literature Index
 LA English
 SL English
 ED Entered STN: 13 Jun 2006
 Last Updated on STN: 13 Jun 2006
 AB Several lines of evidence have provided compelling support for low-density lipoprotein receptor-related protein 5 (LRP5) and the canonical Wnt/beta.-catenin signaling pathway as being important and essential for

bone formation. Lrp5 and its close homolog, Lrp6, are coreceptors with frizzled for Wnt proteins. Binding of Wnt proteins to Lrp5/6 and frizzled activates the Wnt/ β -catenin signaling pathway. Mutations in Lrp5 have been shown to give rise to human diseases of low bone mass and loss of vision such as osteoporosis pseudoglioma syndrome (OPPG) and familial exudative vitreoretinopathy (FEVR) as well as several human conditions with increased bone mass and reduced fracture risk, such as the high bone mass (***HBM***) phenotype. Although it is well established that the Lrp5/6-Wnt canonical pathway is important in embryonic growth and development of the skeleton, its role in the adult skeleton is not clear. Accumulating evidence now supports an important role for Lrp5 in the response of the postnatal skeleton to mechanical load. Transgenic mice carrying the human ***HBM*** mutation (LRP5(G171V)) have increased sensitivity to load, and mice lacking Lrp5 do not respond to mechanical load. In vivo loading of LRP5(G171V) mice tibia results in increased osteoprotegerin (OPG) gene expression. Mice with either gain- or loss-of-function mutations in protein components of the canonical pathway below the level of Lrp5/6 develop high or low bone mass mainly as a consequence of altered OPG production by osteoblasts, which subsequently alters osteodastogenesis. Thus, activation of the canonical Wnt signaling pathway apparently has multiple modes of action on bone cells to regulate bone mass. Given the clear importance of LRP5 in regulating bone mass, this gene/protein represents a potentially exciting new target for the development of anabolic agents to treat osteoporosis.

.COPYRG.
Copyright 2006 by Humana Press Inc. All rights of any nature whatsoever reserved.

L10 ANSWER 2 OF 11 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
DUPLICATE 2
AN 2004:383095 BIOSIS <<LOGINID::20060803>>
DN PREV200400388096
TI High bone mass gene of 1.1q13.3.
AU ***Carulli, John P.*** [Inventor, Reprint Author]; ***Little,***
*** Randall D.*** [Inventor]; ***Recker, Robert R.***
[Inventor];
Johnson, Mark L. [Inventor]
CS ASSIGNEE: Genome Therapeutics Corporation
PI US 6780609 20040824
SO Official Gazette of the United States Patent and Trademark Office Patents,
(Aug 24 2004) Vol. 1285, No. 4.
<http://www.uspto.gov/web/menu/patdata.html>
. e-file.
ISSN: 0098-1133 (ISSN print).
DT Patent
LA English
ED Entered STN: 29 Sep 2004
Last Updated on STN: 29 Sep 2004
AB The present invention relates to methods and materials used to isolate and detect a high bone mass gene and a corresponding wild-type gene, and mutants thereof. The present invention also relates to the high bone mass gene, the corresponding wild-type gene, and mutants thereof. The genes identified in the present invention are implicated in bone development. The invention also provides nucleic acids, including coding sequences, oligonucleotide primers and probes, proteins, cloning vectors, expression vectors, transformed hosts, methods of developing pharmaceutical compositions, methods of identifying molecules involved in bone development, and methods of diagnosing and treating diseases involved in

bone development. In preferred embodiments, the present invention is directed to methods for treating, diagnosing and preventing osteoporosis.

L10 ANSWER 3 OF 11 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
DUPLICATE 3
AN 2004:351355 BIOSIS <<LOGINID::20060803>>
DN PREV200400354810
TI High bone mass gene of 11q13.3.
AU ***Carulli, John P.*** [Inventor, Reprint Author]; ***Little,***
*** Randall D.*** [Inventor]; ***Recker, Robert R.***
[Inventor];
Johnson, Mark L. [Inventor]
CS Southboro, MA, USA
ASSIGNEE: Genome Therapeutics Corporation; Creighton University School of Medicine, Omaha, NE, USA
PI US 6770461 20040803
SO Official Gazette of the United States Patent and Trademark Office Patents,
(Aug 3 2004) Vol. 1285, No. 1.
<http://www.uspto.gov/web/menu/patdata.html>.
. e-file.
ISSN: 0098-1133 (ISSN print).
DT Patent
LA English
ED Entered STN: 26 Aug 2004
Last Updated on STN: 26 Aug 2004
AB The present invention relates to methods and materials used to isolate and detect a high bone mass gene and a corresponding wild-type gene, and mutants thereof. The present invention also relates to the high bone mass gene, the corresponding wild-type gene, and mutants thereof. The genes identified in the present invention are implicated in bone development. The invention also provides nucleic acids, including coding sequences, oligonucleotide primers and probes, proteins, cloning vectors, expression vectors, transformed hosts, methods of developing pharmaceutical compositions, methods of identifying molecules involved in bone development, and methods of diagnosing and treating diseases involved in bone development. In preferred embodiments, the present invention is directed to methods for treating, diagnosing and preventing osteoporosis.

L10 ANSWER 4 OF 11 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
AN 2005:264978 BIOSIS <<LOGINID::20060803>>
DN PREV200510058184
TI Ulna loading response altered by the ***HBM*** mutation.
AU Cullen, D. M. [Reprint Author]; Akhter, M. P.; ***Johnson, M. L.***
Morgan, S.; ***Recker, R. R.***
CS Creighton Univ, Osteoporosis Res Ctr, Omaha, NE USA
SO Journal of Bone and Mineral Research, (OCT 2004) Vol. 19, pp. S396.
Meeting Info.: 26th Annual Meeting of the American-Society-for-Bone-and-Mineral-Research. Seattle, WA, USA. October 01 -05, 2004.
Amer Soc Bone & Mineral Res.
CODEN: JBMREJ. ISSN: 0884-0431.
DT Conference; (Meeting)
Conference; (Meeting Poster)
LA English
ED Entered STN: 21 Jul 2005
Last Updated on STN: 21 Jul 2005

L10 ANSWER 5 OF 11 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN
DUPLICATE 4
AN 2004263068 EMBASE <<LOGINID::20060803>>
TI Bone biomechanical properties in LRP5 mutant mice.
AU Akhter M.P.; Wells D.J.; Short S.J.; Cullen D.M.; ***Johnson M.L.***
Haynatzki G.R.; Babji P.; Allen K.M.; Yaworsky P.J.; Bex F.; ***Recker***
*** R.R.***
CS M.P. Akhter, Osteoporosis Research Center, Creighton University, 601 North, 30th Street #4820, Omaha, NE 68131, United States.
akhtemp@creighton.edu
SO Bone, (2004) Vol. 35, No. 1, pp. 162-169.
Refs: 33

ISSN: 8756-3282 CODEN: BONEDL
 PUI S 8756-3282(04)00084-5
 CY United States
 DT Journal; Article
 FS 029 Clinical Biochemistry
 033 Orthopedic Surgery
 LA English
 SL English
 ED Entered STN: 9 Jul 2004
 Last Updated on STN: 9 Jul 2004
 AB The mutation responsible for the high bone mass (***HBM***) phenotype has been postulated to act through the adaptive response of bone to mechanical load resulting in denser and stronger skeletons in humans and animals. The bone phenotype of members of a ***HBM*** family is characterized by normally shaped bones that are exceptionally dense, particularly at load bearing sites [Cancer Res. 59 (1999) 1572]. The high bone mass (***HBM***) mutation was identified as a glycine to valine substitution at amino acid residue 171 in the gene coding for low-density lipoprotein receptor-related protein 5 (LRP5) [Bone Miner. Res. 16(4) (2001) 758]. Thus, efforts have focused on the examination of the role of LRP5 and the G171V mutation in bone mechanotransduction responses [J. Bone Miner. Res 18 (2002) 960]. Transgenic mice expressing the human G171V mutation have been shown to have skeletal phenotypes remarkably similar to those seen in affected individuals. In this study, we have identified differences in biomechanical (structural and apparent material) properties, bone mass/ash, and bone stiffness of cortical and cancellous bone driven by the G171V mutation in LRP5. As in humans, the LRP5 G171V plays an important role in regulating bone structural phenotypes in mice. These bone phenotypes include greater structural and apparent material properties in ***HBM*** HET as compared to non-transgenic littermates (NTG) mice. Body size and weight in ***HBM*** HET were similar to that in NTG control mice. However, the LRP5 G171V mutation in HET mice results in a skeleton that has greater structural (femoral shaft, femoral neck, tibiae, vertebral body) and apparent material (vertebral body) strength, percent bone ash weight (ulnae), and tibial stiffness. Despite similar body weight to NTG mice, the denser and stiffer bones in G171V mice may represent greater bone formation sensitivity to normal mechanical stimuli resulting in an overadaptation of skeleton to weight-related forces. .COPYRGHT. 2004 Elsevier Inc. All rights reserved.

L10 ANSWER 6 OF 11 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN
 AN 2002346039 EMBASE <<LOGINID::20060803>>
 TI The gene for high bone mass.
 AU ***Johnson M.L.*** ; Picconi J.L.; ***Recker R.R.***
 CS Dr. M.L. Johnson, Osteoporosis Research Center, Creighton Univ. School of Medicine, 601 North 30th Street, Omaha, NE 68131, United States.
 MARKL@creighton.edu
 SO Endocrinologist, (2002) Vol. 12, No. 5, pp. 445-453. .
 Refs: 79
 ISSN: 1051-2144 CODEN: EDOCEB
 CY United States
 DT Journal; General Review
 FS 022 Human Genetics
 033 Orthopedic Surgery
 LA English
 SL English
 ED Entered STN: 17 Oct 2002
 Last Updated on STN: 17 Oct 2002
 AB The mass, density, and architecture of the skeleton are adapted to enable it to perform its mechanical, protective, and metabolic functions.

Osteoporosis is a condition of lost adaptation characterized by decreased skeletal mass and density and increased skeletal fragility. Many diseases result in increased bone density, including osteopetrosis and Paget's disease, but deformities or bony lesions with decreased skeletal integrity usually accompany these conditions. We have identified a kindred with high bone mass (***HBM***) yet normally shaped bones. Linkage analysis localized the gene for the ***HBM*** trait to chromosome 11 (11q12-13). Subsequent physical mapping and mutation analysis have identified the cause as a point mutation in the LDL receptor-related protein 5 (Lrp5) gene that results in a valine substitution for glycine at position 171 in the protein. This protein is important in the Wnt signaling pathway. The authors have hypothesized that the Lrp5 gene/pathway is part of the mechanism by which bone senses mechanical load. Increased bone strength, ***HBM*** , and a phenotype resembling our human kindred develop in transgenic mice carrying the human Lrp5 gene with the ***HBM*** mutation. Recent data indicate that the ***HBM*** mutation reduces the threshold for response of the skeleton to mechanical load resulting in an overadaptation to normal mechanical loads. This discovery has opened the door to understanding one of the most important paradigms in bone biology, how bones respond and adapt to mechanical loading. Understanding the mechanosensation pathway and its regulation will lead us to new treatments for osteoporosis.

L10 ANSWER 7 OF 11 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
 AN 2003:431910 BIOSIS <<LOGINID::20060803>>
 DN PREV200300431910
 TI Bone sensitivity to mechanical loads with the Lrp5 ***HBM*** mutation.
 AU Cullen, D. M. [Reprint Author]; Akhter, M. P. [Reprint Author]; Mace, D. [Reprint Author]; ***Johnson, M. L.*** [Reprint Author]; Babij, P.; ***Recker, R. R.*** [Reprint Author]
 CS Osteoporosis Research Center, Creighton University, Omaha, NE, USA
 SO Journal of Bone and Mineral Research, (September 2002) Vol. 17, No. Suppl 1, pp. S332. print.
 Meeting Info.: Twenty-Fourth Annual Meeting of the American Society for Bone and Mineral Research. San Antonio, Texas, USA. September 20-24, 2002.
 American Society for Bone and Mineral Research.
 ISSN: 0884-0431 (ISSN print).
 DT Conference; (Meeting)
 Conference; (Meeting Poster)
 Conference; Abstract; (Meeting Abstract)
 LA English
 ED Entered STN: 17 Sep 2003
 Last Updated on STN: 17 Sep 2003

L10 ANSWER 8 OF 11 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN
 DUPLICATE 5
 AN 2002013553 EMBASE <<LOGINID::20060803>>
 TI A mutation in the LDL receptor-related protein 5 gene results in the autosomal dominant high-bone-mass trait.
 AU ***Little R.D.*** ; ***Carulli J.P.*** ; Del Mastro R.G.; Dupuis J.; Osborne M.; Folz C.; Manning S.P.; Swain P.M.; Zhao S.C.; Eustace B.; Lappe M.M.; Spitzer L.; Zweier S.; Braunschweiger K.; Benchekroun Y.; Hu X.; Adair R.; Chee L.; Fitzgerald M.G.; Tulig C.; Caruso A.; Tzellas N.; Bawa A.; Franklin B.; McGuire S.; Nogues X.; Gong G.; Allen K.M.; Anisowicz A.; Morales A.J.; Lomedico P.T.; Recker S.M.; Van Eerdewegh P.; ***Recker R.R.*** ; ***Johnson M.L.***
 CS Dr. R.D. Little, Human Genetics Department, Genome Therapeutics

Corporation, 100 Beaver Street, Waltham, MA 02453, United States.
 riittle@genomecorp.com
 SO American Journal of Human Genetics, (2002) Vol. 70, No. 1, pp. 11-19.
 Refs: 32
 ISSN: 0002-9297 CODEN: AJHGAG
 CY United States
 DT Journal; Article
 FS 022 Human Genetics
 LA English
 SL English
 ED Entered STN: 17 Jan 2002
 Last Updated on STN: 17 Jan 2002
 AB Osteoporosis is a complex disease that affects .ltoreq.10 million people in the United States and results in 1.5 million fractures annually. In addition, the high prevalence of osteopenia (low bone mass) in the general population places a large number of people at risk for developing the disease. In an effort to identify genetic factors influencing bone density, we characterized a family that includes individuals who possess exceptionally dense bones but are otherwise phenotypically normal. This high-bone-mass trait (***HBM***) was originally localized by linkage analysis to chromosome 11q12-13. We refined the interval by extending the pedigree and genotyping additional markers. A systematic search for mutations that segregated with the ***HBM*** phenotype uncovered an amino acid change, in a predicted .beta.-propeller module of the low-density lipoprotein receptor-related protein 5 (LRP5), that results in the ***HBM*** phenotype. During analysis of .ltoreq.1,000 individuals from the ***HBM*** kindred. By use of in situ hybridization to rat tibia, expression of LRP5 was detected in areas of bone involved in remodeling. Our findings suggest that the ***HBM*** mutation confers a unique osteogenic activity in bone remodeling, and this understanding may facilitate the development of novel therapies for the treatment of osteoporosis.

L10 ANSWER 9 OF 11 CAPLUS COPYRIGHT 2006 ACS on STN
 AN 2001:886643 CAPLUS <<LOGINID::20060803>>
 DN 136:32816
 TI Regulating lipid levels via the human Zmax1 or high-bone-mass ***HBM*** gene
 IN ***Carulli, John P.*** ; ***Little, Randall D.*** ; ***Recker,***
 *** Robert R.*** ; ***Johnson, Mark L.***
 PA Genome Therapeutics Corporation, USA; Creighton University School of Medicine
 SO PCT Int. Appl., 409 pp.
 CODEN: PIXXD2
 DT Patent
 LA English
 FAN.CNT 4

PATENT NO.	KIND	DATE	APPLICATION NO.
PI WO 2001092891	A2	20011206	WO 2001-US16946
20010525			
WO 2001092891	A3	20020725	
W:	AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HU, HR, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW		
RW:	GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG		
CA 2410253	AA	20011206	CA 2001-2410253
20010525			

AU 2001269712 A2 20011211 AU 2001-269712
 20010525
 EP 1285002 A2 20030226 EP 2001-948240
 20010525
 R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR
 BR 2001011057 A 20030415 BR 2001-11057
 20010525
 JP 2004523724 T2 20040805 JP 2002-501047
 20010525
 NZ 522600 A 20040924 NZ 2001-522600
 20010525
 ZA 2002008977 A 20031105 ZA 2002-8977
 20021105
 PRAI US 2000-578900 A 20000526
 WO 2001-US16946 W 20010525
 AB The present invention relates to the high bone mass (***HBM***) gene, the corresponding wild-type gene (Zmax1), and mutants thereof. The Zmax1/ ***HBM*** gene was located on chromosome 11q13.3 by genetic linkage and mutation anal. Cloning methods using bacterial artificial chromosomes enabled focus on the chromosome region of 11q13.3 and sequencing of the autosomal dominant gene. A guanine-to-thymine polymorphism at position 582 (glycine-to-valine at position 171 in the protein) in Zmax1 gene produces the ***HBM*** gene and the ***HBM*** phenotype as well as altered lipid levels. Hybridization for Zmax1 is primarily detected in areas of bone involved in remodeling, including the endosteum and trabecular bone within the metaphysis; pos. signals are also obsd in selected bone lining cells of the periosteum and epiphysis and in chondrocytes within the growth plate. The genes identified in the present invention are implicated in regulation of physiol. lipid levels, and thereby lipid-mediated diseases and conditions. The invention also provides nucleic acids, including coding sequences, oligonucleotide primers and probes, proteins, cloning vectors, expression vectors, transformed hosts, methods of developing pharmaceutical compns., methods of identifying mols. involved in lipid level regulation in a subject. In preferred embodiments, the present invention is directed to methods for treating and preventing atherosclerosis, arteriosclerosis cardiovascular disease, atherosclerotic and arteriosclerotic assocd. conditions.

L10 ANSWER 10 OF 11 CAPLUS COPYRIGHT 2006 ACS on STN
 AN 2001:763189 CAPLUS <<LOGINID::20060803>>
 DN 135:328141
 TI Human gene Zmax1 of 11q13.3, ***HBM*** (high bone mass) allele, encoded polypeptides, and their diagnostic and therapeutic uses
 IN ***Carulli, John P.*** ; ***Little, Randall D.*** ; ***Recker,***
 *** Robert R.*** ; ***Johnson, Mark L.***
 PA Genome Therapeutics Corporation, USA
 SO PCT Int. Appl., 443 pp.
 CODEN: PIXXD2
 DT Patent
 LA English
 FAN.CNT 4

PATENT NO.	KIND	DATE	APPLICATION NO.
PI WO 2001077327	A1	20011018	WO 2000-US16951
20000621			
W:	AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW		
RW:	GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY,		

DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
 US 6770461 B1 20040803 US 2000-544398
 20000405
 US 6780609 B1 20040824 US 2000-543771
 20000405
 CA 2402410 AA 20011018 CA 2000-2402410
 20000621
 EP 1268775 A1 20030102 EP 2000-941578
 20000621
 R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL
 JP 2004515209 T2 20040527 JP 2001-575181
 20000621
 NZ 521769 A 20041224 NZ 2000-521769
 20000621
 US 2003219793 A1 20031127 US 2003-374979
 20030228
 PRAI US 2000-543771 A 20000405
 US 2000-544398 A 20000405
 US 1998-71449P P 19980113
 US 1998-105511P P 19981023
 US 1999-229319 A2 19990113
 US 2000-578900 W 20000526
 WO 2000-US16951 W 20000621
 US 2002-240851 A1 20021004
 AB The present invention relates to methods and materials used to isolate and detect a high bone mass gene and a corresponding wild-type gene, and mutants thereof. The present invention also relates to the high bone mass allele, the corresponding wild-type gene, Zmax1, and mutants thereof. The genes identified in the present invention are implicated in bone development and in focal adhesion signaling. The invention also provides nucleic acids, including coding sequences, oligonucleotide primers and probes, proteins, cloning vectors, expression vectors, transformed hosts, methods of developing pharmaceutical compns., methods of identifying mols. involved in bone development, and methods of diagnosing and treating diseases involved in bone development. In preferred embodiments, the present invention is directed to methods for treating, diagnosing and preventing osteoporosis. The invention describes expanded pedigree anal. and genetic linkage anal. of a high bone mass (***HBM***) gene now known as an allele of human gene Zmax1. Older individuals with the ***HBM*** allele do not show loss of bone mass compared to normal individuals, do not have osteoporosis, and do not have any known high bone mass syndrome. Gene Zmax1 was localized between genetic markers on human chromosome 11q13.3 and subsequently, BAC clones with the gene were sequenced. The ***HBM*** allele is inherited as an autosomal dominant gene and is a G.fwdarw. T mutation at nucleotide 582 in exon 3 which results in a G171V substitution in the encoded protein. Addnl. genotyping of 911 individuals established that the ***HBM*** allele is rare and never found in unaffected individuals. "Silent" SNPs (single nucleotide polymorphisms) in the gene Zmax1 region were also identified. Gene Zmax1 encodes an LDL-receptor-related protein and the ***HBM*** mutation occurs in a conserved region of the presumed extracellular domain. Proteins interacting with the cytoplasmic domain of gene Zmax1 protein in a yeast two-hybrid assay were identified and include many proteins found at cell-cell and cell-matrix contact sites. These results suggest a potential role for gene Zmax1 in focal adhesion signaling and suggest that regulating gene Zmax1 expression or protein binding may affect bone processes.
 RE.CNT 12 THERE ARE 12 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 11 OF 11 EMBASE COPYRIGHT (c) 2006 Elsevier
 B.V. All rights reserved on STN
 DUPLICATE 6
 AN 97183437 EMBASE <<LOGINID::20060803>>
 DN 1997183437
 TI Linkage of a gene causing high bone mass to human chromosome 11 (11q12-13).
 AU ***Johnson M.L.*** ; Gong G.; Kimberling W.; Recker S.M.; Kimmel D.B.;
 Recker R.R.
 CS Dr. R.R. Recker, Osteoporosis Research Center, Creighton University, 601 North 30th Street 4820, Omaha, NE 68131-5149, United States. rrecker@creighton.edu
 SO American Journal of Human Genetics, (1997) Vol. 60, No. 6, pp. 1326-1332.
 Refs: 29
 ISSN: 0002-9297 CODEN: AJHGAG
 CY United States
 DT Journal; Article
 FS 022 Human Genetics
 LA English
 SL English
 ED Entered STN: 10 Jul 1997
 Last Updated on STN: 10 Jul 1997
 AB The purpose of this paper is to report the linkage of a genetic locus (designated ' ***HBM*** ') in the human genome to a phenotype of very high spinal bone density, using a single extended pedigree. We measured spinal bone mineral density, spinal Z(BMD), and collected blood from 22 members of this kindred. DNA was genotyped on an Applied Biosystems model 377 (ABI PRISM Linkage Mapping Sets; Perkin Elmer Applied Biosystems), by use of fluorescence-based marker sets that included 345 markers. Both two-point and multipoint linkage analyses were performed, by use of affected/unaffected and quantitative-trait models. Spinal Z(BMD) for affected individuals (N = 12) of the kindred was 5.54 +/- 1.40; and for unaffected individuals (N = 16) it was 0.41 +/- 0.81. The trait was present in affected individuals 18-86 years of age, suggesting that ***HBM*** influences peak bone mass. The only region of linkage was to a series of markers on chromosome 11 (11q12-13). The highest LOD score (5.21) obtained in two-point analysis, when a quantitative-trait model was used, was at D11S987. Multipoint analysis using a quantitative-trait model confirmed the linkage, with a LOD score of 5.74 near marker D11S987. ***HBM*** demonstrates the utility of spinal Z(BMD) as a quantitative bone phenotype that can be used for linkage analysis. Osteoporosis pseudoglioma syndrome also has been mapped to this region of chromosome 11. Identification of the causal gene for both traits will be required for determination of whether a single gene with different alleles that determine a wide range of peak bone densities exists in this region.
 =>
 ---Logging off of STN---
 =>
 Executing the logoff script...
 => LOG Y
 COST IN U.S. DOLLARS
 FULL ESTIMATED COST
 SINCE FILE ENTRY
 74.28
 TOTAL SESSION
 74.49

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)
SINCE FILE TOTAL

	ENTRY	SESSION
CA SUBSCRIBER PRICE	-4.50	-4.50

STN INTERNATIONAL LOGOFF AT 16:12:19 ON 03 AUG 2006